1.05 +/−0.11mm and 1.24 +/−0.09mm in the 3-minute time group (p NS between groups). Intimal hyperplasia was modest in all rats, with no significant differences in intimal wall areas. The intimal and medial wall area as a percentage of overall vessel area was 35.0 +/−5.2% in the 30-second group and 36.8 +/−6.6% in the 3-minute group (p NS).

Conclusions: Prolonged balloon inflation time failed to produce significant changes in intimal disruption after 24 hours. At 30 days, there was no difference in the amount of intimal hyperplasia, medial wall thickening, or lumen diameter compared to standard inflation times. These results do not support changing current balloon angioplasty practices.

Author Disclosures: D. Boone: Nothing to disclose; **G. Jour**: Nothing to disclose; **J. C. Lantis**: Nothing to disclose; **G. S. Schwartz**: Nothing to disclose.

C3j: Poster Session - Research (2)

PS218.

Inhibition of CXCL4-CCL5 Chemokine Interaction Ameliorates the Development of Abdominal Aortic Aneurysm in Mice

Yasunori Iida[1], Baohui Xu[1], Joshua Schultz[2], Court R. Turner[2], Ronald L. Dalman[1]. [1]Vascular Surgery, Stanford University School of Medicine, Stanford, CA; [2]Carolus Therapeutics, Inc., La Jolla, CA

Objectives: Abdominal aortic aneurysm (AAA) is a macrophages-driven vascular disease. Given the importance of CXCL4-CCL5 chemokine interaction in macrophage/monocytes migration into inflamed arteries, we evaluated the effect of a novel synthetic peptide inhibitor to CXCL4-CCL5 interaction on AAA progression in the porcine pancreatic elastase (PPE)-induced AAA mouse model.

Methods: AAA was created in 10-wk-old male C57BL/6 mice by PPE infusion into infrarenal aorta. These mice were injected i.v. with the synthetic peptide inhibitor to CXCL4-CCL5 interaction (CT-2009ca, 10 (n=5) or 20 (n=7) mg/kg/day or the vehicle (n=8) (Carolus Therapeutics, Inc) for 3 days before PPE infusion and 13 days thereafter. The AAA was imaged by ultrasonography and defined as a 50% increase in the aortic diameter. The aortae were harvested 14 days after PPE infusion and subjected to histopathology.

Results: AAA developed in all mice treated with the vehicle within 7 days. In contrast, AAA developed in 3 mice and 1 mouse treated with the CT-2009ca at the dose of 10 and 20 mg /kg/day, respectively, within 14 days. The CT-2009ca treatment dose-dependently inhibited the PPE-induced increase in aorta diameter. Moreover, the elastic Masson staining and the anti-smooth muscle cell alpha actin antibody immunostaining revealed that medial elastin fibers and smooth muscle cell layers were well preserved in the CT-2009ca-treated mice as compared to the vehicle-treated mice. As expected, the CT-2009ca treatment significantly reduced the numbers of macrophages, CD4+ T cells and neutrophils and newly formed blood vessels in the media and adventitia by immunostaining with the mAbs against leukocyte subsets and endothelial cells.

Conclusions: In the mouse PPE model of AAA, inhibition of CXCL4-CCL5 interaction by CT-2009ca suppressed AAA development by reducing angiogenesis and limiting medial and adventitial infiltration of certain subsets of leukocytes. Thus, CT-2009ca may be a potential therapeutic candidate for treating human AAA disease.

Author Disclosures: R. L. Dalman: Nothing to disclose; **Y. Iida**: Nothing to disclose; **J. Schultz**: Nothing to disclose; **C. R. Turner**: Nothing to disclose; **B. Xu**: Nothing to disclose.

PS220.

Thermo-Mechanical Resistance of a Nanocomposite Polymer Exposed to Simulated in Vivo Hydrodynamic Fatigue for Ten Years in Development of a Sutureless Endovascular Stent Graft

Mital Y. Desai[1], Raheleh Bakhshi[2], Arnold Darbyshire[2], Max Ahmed[2], James Eaton-Evans[3], Xiang Zhou[3], Zhong You[3], Alexander M. Seifalian[2], George Hamilton[1]. [1]Vascular Surgery, Royal Free Hampstead NHS Trust, London, United Kingdom; [2]Division of Surgery and Interventional Science, University College London, London, United Kingdom; [3]Department of Engineering Science, University of Oxford, Oxford, United Kingdom

Objectives: To physiologically test thermo-mechanical resistance of a nanocomposite polymer based on polyhedral oligomeric silsesquioxane (POSS) and poly (carbonate-urea) urethane (PCU) to assess its feasibility as graft material for a sutureless aortic stent-graft by exposing it to simulated in vivo hydrodynamic fatigue according to FDA guidelines.

Methods: Aortic stent-grafts (n=4) were tested in 37 degree Celsius distilled water using in vitro accelerated circumferential pulsatile model. After analysis for 400-million cycles equivalent to 10-years in human body, the mechanical and elastic properties were determined by Fourier transform infrared spectroscopy (FTIR), radial stress-strain studies, and linear elasticity tests. Dynamic scanning calorimetry (DSC) and Thermo-mechanical analysis (TMA) were used to assess thermal resistance. Comparison was made with zero-cycled control.

Results: All stent-grafts (n=4; diameter 25±0.1 mm; length 25.3±0.5 cm) successfully completed accelerated pulsatile fatigue testing at 37°C and pulse pressure 94±14 mm Hg over 12 months. FTIR showed increased intensity of -NHCO- bonds (62% transmittance in tested specimen compared to 47% in control) but there was no difference in intensity of other major bonds and no significant sign of degradation. Tensile strain of fatigue-tested polymer compared favourably with zero-cycled control at 50% to 500% stress (P=0.69). When stretched up to 500% original

length, there was no difference in linear elasticity (deformation 13.3% in tested specimen compared to 12.3% in control; P=0.74). DSC and TMA showed decrease in glass transition temperature up to 4°C but thermo-tropic transition was comparable to control.

Conclusions: Simulated physiological in vivo hydrodynamic fatigue has no significant degradative effect on an innovative stent-graft from POSS-PCU nanocomposite polymer. Sutureless technology incorporating nitinol stents proved to be robust with no separation over accelerated 10-year cycle.

Author Disclosures: M. Ahmed: Nothing to disclose; **R. Bakhshi**: Nothing to disclose; **A. Darbyshire**: Nothing to disclose; **M. Y. Desai**: Nothing to disclose; **J. Eaton-Evans**: Nothing to disclose; **G. Hamilton**: Nothing to disclose; **A. M. Seifalian**: Nothing to disclose; **Z. You**: Nothing to disclose; **X. Zhou**: Nothing to disclose.

PS222.

Percutaneous Peri-Adventitial Guanethidine Delivery Induces Renal Artery Sympathectomy: Preclinical Experience and Implication for Refractory Hypertension

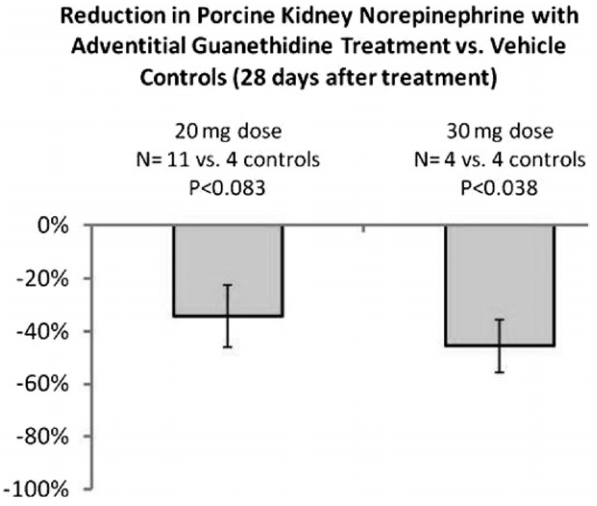
Christopher Owens[1], Warren J. Gasper[1], Kirk Seward[3], Sanjay Misra[4], Stewart B. Jacobson[2], Russell M. Jones[2]. [1]University of California San Francisco, San Francisco, CA; [2]CVPath, Gaithersburg, MD; [3]Mercator MedSystems, San Leandro, CA; [4]Mayo Clinic, Rochester, MN

Objectives: Renal efferent sympathetic nerve overactivity is contributory to the hypertensive state. Guanethidine is known to induce an autonomic denervation through an immune mediated pathway.We hypothesized that guanethidine could be safely delivered into the renal perivascular space to produceautonomic denervation and reduction in renal norepinephrine (NE) content.

Methods: A micro-infusion catheter (Mercator MedSystems) was introduced via the femoral artery into the renal arteries of (N=11) female swin.Two to three ml of guanethidine (20-30 mg) or vehicle, each with 20% dilution of contrast, was infused into the adventitial space under fluoroscopic guidance. The primary efficacy endpoint in this study was kidney NE tissue content measured by high-performance liquid chromatography.Secondary outcomes included histological evidence of renal artery denervation to corroborate efficacy and renal artery injury and neural inflammation scores.

Results: There were 18 treated renal arteries and 4 vehicle controls in this study.There was 100% procedural success in the delivery of guanethidine or vehicle to the renal artery adventitial space.The mean renal artery injury scores were not different between treated and control pigs (P=NS), and in all cases the endothelial integrity was maintained.However,in all guanethidine-treated cases there was histological evidence of peri-neural inflammation and nerve destruction whereas the nerves appeared normal in all control animals.There was an overall decrease in renal cortex NE content in a dose dependent fashion, control 245±34 ng/g tissue, 20 mg dose 161±18 ng/g tissue and 30 mg dose 133±16 ng/g tissue, P=.038 (Figure).

Conclusions: These data support the hypothesis that guanethidine can be delivered safely and efficiently into the renal peri-adventitial space through a novel minimally invasive technique.Further, renal NE content is significantly reduced suggesting that this could be a viable procedure in the treatment of refractory sympathetic-driven hypertension.



Author Disclosures: W. J. Gasper: Nothing to disclose; **S. B. Jacobson**: Nothing to disclose; **R. M. Jones**: Nothing to disclose; **S. Misra**: Nothing to disclose; **C. Owens**: Nothing to disclose; **K. Seward**: Mercator MedSystems, Ownership or PartnershipR43HL102998, Research Grants.

PS224.

Porcine Mesenchymal Stem Cell Labeling with MRI Contrast Agent Ferex

Mehrnoosh Shahrivar[1], Daniel K. Han[1], Karen Briley-Saebo[2], Andrew Tye[1], Michael L. Marin[1], Peter L. Faries[1]. [1]Division of Vascular Surgery, Department of Surgery, Mount Sinai School of Medicine, New York, NY; [2]Department of Radiology, Mount Sinai School of Medicine, New York, NY

Objectives: Mesenchymal stem cell (MSC) transplantation is currently being investigated in porcine abdominal aortic aneurysm (AAA) models for its potential as regenerative treatment. Reliable methods of labeling and tracking MSCs are necessary to evaluate their effects. This study aims to evaluate in vitro performance of Ferex, a superparamagnectic iron oxide that can be tracked by magnetic